

Electromyogenic Artifacts and Electroencephalographic Inferences

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Abstract Muscle or electromyogenic (EMG) artifact poses a serious risk to inferential validity for any electroencephalography (EEG) investigation in the frequency-domain owing to its high amplitude, broad spectrum, and sensitivity to psychological processes of interest. Even weak EMG is detectable across the scalp in frequencies as low as the alpha band. Given these hazards, there is substantial interest in developing EMG correction tools. Unfortunately, most published techniques are subjected to only modest validation attempts, rendering their utility questionable. We review recent work by our laboratory quantitatively investigating the validity of two popular EMG correction techniques, one using the general linear

model (GLM), the other using temporal independent component analysis (ICA). We show that intra-individual GLM-based methods represent a sensitive and specific tool for correcting on-going or induced, but not evoked (phase-locked) or source-localized, spectral changes. Preliminary work with ICA shows that it may not represent a panacea for EMG contamination, although further scrutiny is strongly warranted. We conclude by describing emerging methodological trends in this area that are likely to have substantial benefits for basic and applied EEG research.

Keywords EMG · EEG · ERSP · ERS/ERD · LORETA source localization · Review

This article is one of five on the “Special Topic: Discussing Gamma” in issue 21(3) of *Brain Topography*.

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Separating neural signals from biological artifacts is a ubiquitous problem for any neurophysiological investigation. Biological artifacts can compromise sensitivity or masquerade as genuine effects, particularly when they are confounded with statistical contrasts. When such artifacts are markedly smaller than neural signals, they can be safely ignored. When they are rare, they may be rejected with trivial consequences. Here we review methods designed to correct a particular biological artifact—muscle or electromyogenic (EMG) activity—that is neither small nor rare.

Peak cranial EMG is ~ 1 – 2 orders of magnitude larger than typical mean differences in the EEG (75 – 400 μV vs. < 10 μV), indicating that even modest contamination can seriously distort inferences. More importantly, because facial EMG is sensitive to a variety of cognitive and affective processes (Tassinary et al. 2007), it is often temporally confounded with experimental manipulations. Consequently, EMG poses a serious risk to inferential validity for investigations of ongoing, induced or evoked EEG in the frequency-domain.

Nature of the EMG Problem

Separation of any two biological signals—in this case myogenic from neurogenic—depends on the degree to which they can be discriminated on one or more dimensions: temporal, anatomical, or spectral. Unfortunately, in addition to co-occurring in time, EMG and EEG possess broadly overlapping anatomical and spectral profiles. EMG arises from muscles across the cranium (Fig. 1a). Because of volume conduction, even weak EMG (Fig. 1b) is detectable across the scalp (Goncharova et al. 2003) in frequencies at least as low as the alpha (8–13 Hz) band (ibid).

Furthermore, EMG exhibits less stereotypy than other biological artifacts. Ocular and cardiac artifacts, for example, arise from fixed sources and do not fundamentally differ

across individuals. EMG, on the other hand, arises from the activity of spatially distributed, functionally independent muscle groups, each with a distinct spectral signature (Goncharova et al. 2003). The relative contributions of each to observed cranial EMG shows substantial variability across individuals and elicitors (Tassinary et al. 2007). These observations indicate that canonical spatial or spectral filters cannot be used to correct EMG artifact. Nevertheless, for work employing high-density arrays, excluding sensors situated at the edge of the montage from analyses may provide a minimal degree of protection from gross EMG and other kinds of artifact. Because such sensors typically provide the clearest measure of such artifacts, they should only be dropped after the application of artifact correction techniques (e.g., regression, blind source separation).

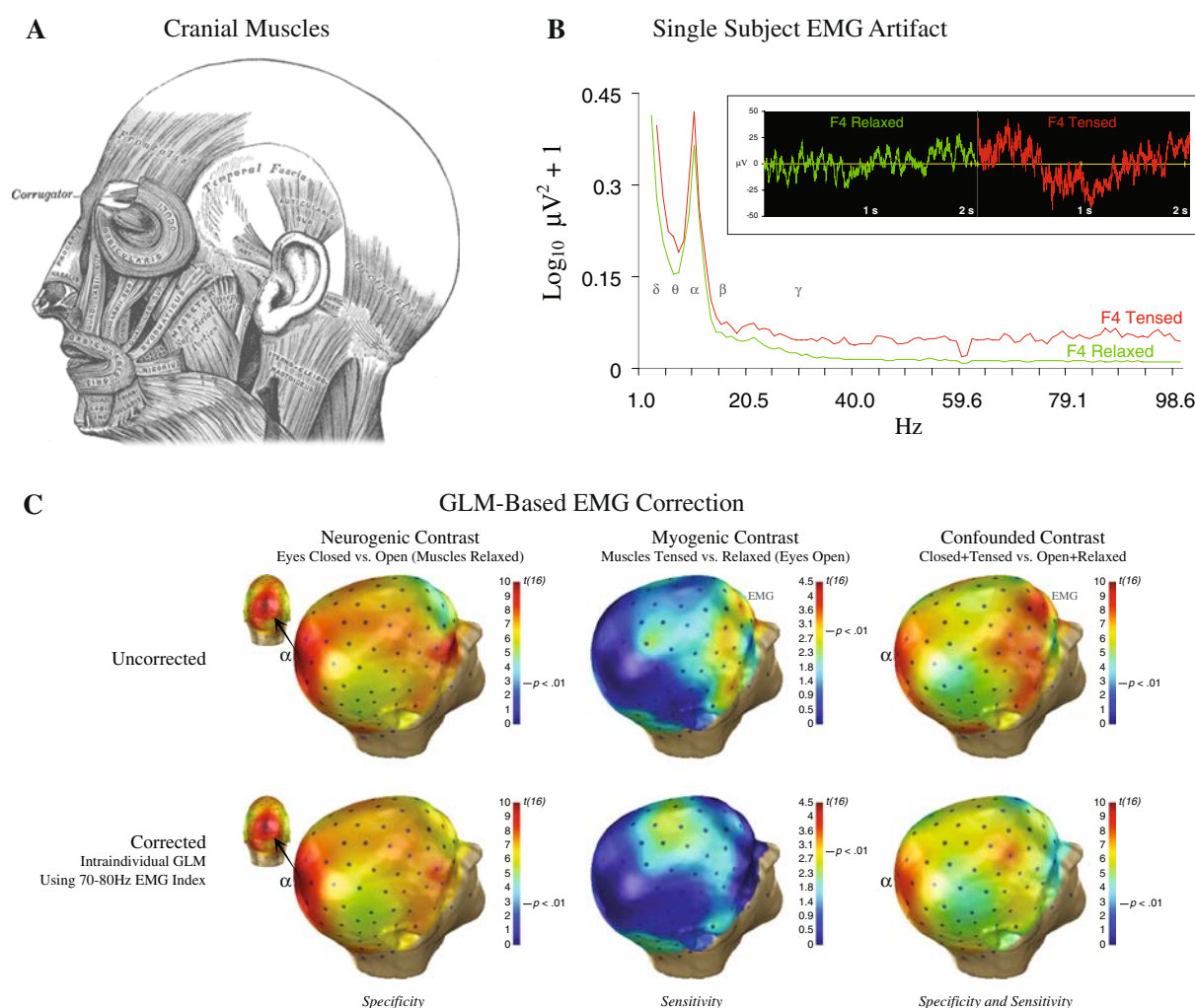


Fig. 1 EMG and EEG. **a** Cranial muscles. **b** EMG artifact at right mid-frontal electrode, F4. Main panel: Power densities during muscle tensing/relaxation. EEG bands denoted by greek symbols. Inset: Single-subject time-series after gross artifact rejection, prior to EMG correction. **c** Group ($n = 17$) results for the alpha band before (upper row) and after (bottom row) intra-individual, GLM-based EMG

correction (adapted from McMenamin et al. in press). Neurogenic alpha activity at parieto-occipital sensors denoted by “ α ”. Myogenic alpha activity at prefrontal sensors denoted by “EMG.” Intra-individual GLM-based EMG correction preserved neurogenic activity in the absence of EMG (“specificity”) and attenuated EMG in the absence of neurogenic activity (“sensitivity”)

Validating EMG Correction Tools

Given the clear inferential hazards posed by EMG, there is great interest in developing tools to separate myogenic from neurogenic signals. Unfortunately, most published techniques are subjected to only modest attempts at validation (McMenamin et al. in press). Ideally, validation would quantitatively establish that a technique possesses a high degree of sensitivity (i.e., attenuates myogenic artifact) and specificity (i.e., preserves neurogenic signals) in a reasonably large and varied sample.

Quantitatively establishing sensitivity and specificity requires data in which the presence and absence of EMG is definitive or can be reasonably assumed. Typically, this involves either simulations, in which EMG is “injected” into otherwise artifact-free EEG (Crespo-Garcia et al. 2008; De Clercq et al. 2005; Delorme et al. 2007; Fitzgibbon et al. 2007), or scripted data, in which participants alternate tensing and relaxing in response to instructions (McMenamin et al. in press). The two methods possess complementary assumptions. The advantage of simulations is that the presence of EMG and stability of neurogenic activity is definitive. The disadvantage is that the assumptions underlying “injection” may not characterize real EMG contamination, biasing the results in favor of techniques that make similar assumptions (Grouiller et al. 2007). The advantage of scripted data is the absence of that assumption. The disadvantage is the need to assume that EMG is absent during periods of quiescence, and that neurogenic activity is consistent across periods of tensing and quiescence. A potential limitation of both techniques is their assumption that myogenic activity makes a negligible contribution to the EEG band of interest during periods of quiescence (cf. Fig. 1b). Depending on the band (e.g., gamma vs. alpha) and degree of compliance with instructions, this assumption may not be tenable (Whitham et al. 2008, 2007).

EMG Correction for High-Density EEG

General Linear Model (GLM)

One tool for correcting EMG artifacts involves using variants of the GLM (e.g., regression, ANCOVA) to remove variance in a neurogenic band of interest (e.g., alpha) that is predicted by activity in an EMG (e.g., 70–80 Hz) band. The advantage of this technique is that it is automatic and, by performing separate corrections at each site, can accommodate anatomical variation. Also, it does not require dedicated EMG channels. While useful for ongoing and induced EEG investigations (Lutz et al. 2004), it does not allow time-series reconstruction, limiting its usefulness for event-related spectral perturbation (ERSP) analyses.

We recently tested this technique’s validity using scripted data (McMenamin et al. in press). High-density EEG data (125-channel; $n = 17$) were acquired while neurogenic and myogenic activation was independently varied by crossing an alpha-blocking manipulation (i.e., eyes opened/closed) with low-intensity muscle activation (i.e., tensing/quiescence). Gross artifacts were rejected before correction, yielding a more realistic degree of contamination (Fig. 1b). Inspection of the uncorrected data revealed that the peak and topography of the alpha-blocking contrast was markedly disturbed when changes in EMG and EEG covaried (Fig. 1c).

We then examined the sensitivity and specificity of inter- and intra-individual variants of the GLM technique for correcting alpha-band neurogenic activity. Sensitivity was quantified by comparing “corrected” EMG-contaminated data to uncorrected EMG-free data in an anterior, myogenic region of interest (ROI). Equivalence tests (Seaman and Serlin 1998) were used as a follow-up. Specificity was similarly quantified using a posterior, neurogenic (i.e., alpha-blocking) ROI.

Results showed that only intra-individual correction, which models correlations between EEG and EMG bands across 1.024-s segments separately for each participant ($df_{\text{observed}} = \text{number of segments} - 2$), showed adequate performance (Fig. 1c). Unfortunately, parallel analyses on source-localized data indicated that none of the GLM-based techniques, when applied in a voxelwise manner, adequately corrected the data.

Independent Component Analysis (ICA)

A second tool for correcting EMG artifacts exploits ICA (Delorme et al. 2007) to blindly separate each individual’s EEG into temporally independent components. Using manual or algorithmic classification (ibid; Fitzgibbon et al. 2007; Mammone and Morabito in press), some components are classified as EMG and discarded prior to reconstruction of the corrected time-series. Typically, this requires inspection of as many components as Channels \times Participants, although principal components analysis or other means (Li et al. 2008) can be used to reduce dimensionality prior to ICA (Hu et al. 2005). To date, ICA has been subjected to only modest attempts, using simulated or ad hoc data, at validation. Moreover, few investigators provide detailed descriptions of their IC classification protocol or estimates of inter-rater reliability (Fatourechi et al. 2007).

Superficially, the question of classifying an IC as EMG appears trivial—it is whatever “looks like EMG” in the temporal (“fuzzy”-looking traces), anatomical (broad fringe/rim distribution; isolated channel, suggesting superficial source), and spectral (broad, high-frequency peak; accelerating/flat spectrum) domains. But in our own

experience, this question is of the utmost importance (cf. Fitzgibbon et al. 2007).

To illustrate this problem, we describe an ad hoc validation test conducted on unpublished 105-channel ERSP data time-locked to 100 ms face presentations. The first 40 independent components (92% variance) were manually classified as (i) EEG, (ii) “pure” EMG, (iii) a mixture, or (iv) non-EMG artifacts (Fig. 2a). Correction was performed twice: first removing only non-EMG artifacts, and then removing both non-EMG and pure EMG artifacts (i.e., mixed EMG/EEG components were retained).

Because EMG was not explicitly manipulated in this experiment, validation required another means of identifying data contaminated by greater or lesser amounts of myogenic activity. Accordingly, we exploited individual differences in EMG to create two groups of participants. The high-EMG group ($n = 22$) displayed two or more pure EMG components, whereas the low-EMG group ($n = 24$)

displayed one or none. Consistent with expectation, confirmatory analyses indicated that the high-EMG group showed more EMG contamination before ICA correction. Specifically, the cumulative variance predicted by the pure EMG components was greater for the high-EMG ($M_s = 0.48$, 2%) than the low-EMG group ($M_s = 0.19$, 0.19%), $P_s < .001$. This effect was specific to the pure EMG components; the variance predicted by components classified as non-EMG artifact ($P = .99$) and mixed EMG/EEG ($P = .12$) did not differ. Not surprisingly, the Group \times Component-Type (Pure EMG, Mixed EMG/EEG) contrast was also significant, $P < .001$. As a coarse test of ICA’s sensitivity, we then performed ERSP analyses comparing activity in the gamma band (25–50 Hz) across groups. Notably, this revealed that the high-EMG group displayed greater power (Fig. 2b, c) over lateral-frontal sites (~ 300 –400 ms) before and after ICA-based EMG correction.

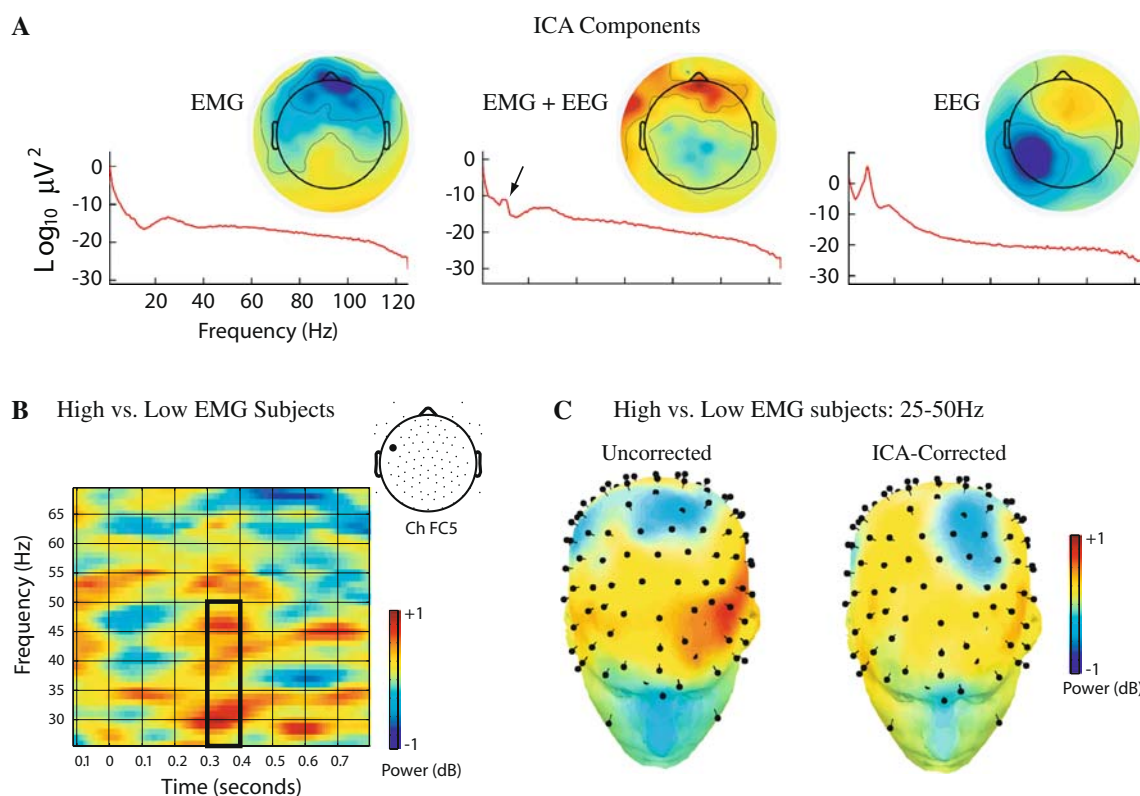


Fig. 2 ICA-based EMG correction. **a** Power spectra and back-projected scalp topographical maps for representative independent components. Note that the polarity of the maps is arbitrary, with scaling proportional to μV . On the scalp, the back-projected myogenic component (“EMG”) exhibits peak activity at midline frontopolar sensors; the neurogenic component (“EEG”) manifests as a broad anterior–posterior dipole, peaking in the vicinity of the left mastoid; and the mixed-source component (“EMG + EEG”) manifests as localized clusters of activity at sensors along the edge of the face, superimposed upon more broadly distributed posterior midline activity. Arrow indicates a peak in the alpha band, indicative of

neurogenic activity. **b** Box indicates that high-EMG subjects exhibited greater activity than low-EMG subjects in the 25–50 Hz gamma band (300–400 ms post-stimulus) at channel FC5 (location shown in inset) after ICA-based EMG correction. As detailed in the text, this group difference in evoked “gamma” activity likely reflects residual myogenic activity. **c** Spline-interpolated whole-head maps of the group mean difference for the 25–50 Hz band. “Pins” denote electrodes. The High-EMG group shows greater activity (shown in red) at the anterior edge of the high-density array before (“Uncorrected”) and, to a lesser degree, after (“ICA-Corrected”) EMG correction

Collectively, our results suggest that the group difference in gamma band ERSP activity following correction reflects residual EMG artifact. Two mechanisms, both entailing inadequate separation of EMG from EEG, might account for this. It could be that undercorrection reflects the decision to retain mixed EMG/EEG components, which non-significantly accounted for one-third more variance in the high-EMG ($M = 2.16\%$) than low-EMG group ($M = 1.63\%$). Alternatively, our putatively pure EEG components may have contained subtle EMG.

These observations raise several important questions. In particular, the optimal number of components to extract and classify remains unknown. This decision is likely to have a marked impact on the quality of source separation. Likewise, the appropriate classification of components containing varying ratios of neurogenic and myogenic activity, and the impact of removing such mixed-source components on sensitivity and specificity is unresolved. We (McMenamin et al. in preparation) are currently pursuing the answers to these questions by pairing the data and validation methods of McMenamin et al. (in press) with ICA-based source separation.

For now, our preliminary findings and other investigators' work (Fitzgibbon et al. 2007) demonstrate that ICA does not necessarily provide adequate protection against EMG artifact. They also highlight the substantial impact that the choice of component classification and rejection protocol can have on inferential validity.

Future Challenges

By describing the challenges of correcting EMG, we hope to stimulate the development and validation of more powerful tools. Promising techniques include using other separation algorithms (Fitzgibbon et al. 2007), incorporating more dimensions (e.g., spatial and spectral) in the decomposition, using higher statistical moments for component classification (Mammone and Morabito in press), and incorporating source anatomy (Barbati et al. 2008). Another promising direction is to validate prospective correction techniques using data obtained in the presence and absence of neuromuscular blockade (cf. Whitham et al. 2008, 2007). Doing so would avoid the assumptions required by simulated and scripted datasets.

Conclusions

Recent years have witnessed a renaissance of interest in using scalp-recorded and source-localized EEG to answer fundamental questions about how mind arises from brain (Makeig et al. 2004; Pizzagalli 2007) and exploiting this

knowledge to interface brains with computers (Fatourechhi et al. 2007). The development and careful validation of novel tools for separating myogenic from neurogenic signals will have substantial benefits for both endeavors.

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References

- Barbati G, Porcaro C, Hadjipapas A, Adjamian P, Pizzella V, Romani GL, Seri S, Tecchio F, Barnes GR (2008) Functional source separation applied to induced visual gamma activity. *Hum Brain Mapp* 29:131–141
- Crespo-Garcia M, Atienza M, Cantero JL (2008) Muscle artifact removal from human sleep EEG by using independent component analysis. *Ann Biomed Eng* 36:467–475
- De Clercq W, Vergult A, Vanrumste B, Van Hees J, Palmieri A, Van Paesschen W, Van Huffel S (2005) A new muscle artifact removal technique to improve the interpretation of the ictal scalp electroencephalogram. *Conf Proc IEEE Eng Med Biol Soc* 1:944–947
- Delorme A, Sejnowski T, Makeig S (2007) Enhanced detection of artifacts in EEG data using higher-order statistics and independent component analysis. *Neuroimage* 34(4):1443–1449
- Fatourechhi M, Bashashati A, Ward RK, Birch GE (2007) EMG and EOG artifacts in brain computer interface systems: a survey. *Clin Neurophysiol* 118:480–494
- Fitzgibbon SP, Powers DMW, Pope KJ, Clark CR (2007) Removal of EEG noise and artifact using blind source separation. *J Clin Neurophysiol* 24:232–243
- Goncharova II, McFarland DJ, Vaughan TM, Wolpaw JR (2003) EMG contamination of EEG: spectral and topographical characteristics. *Clin Neurophysiol* 114:1580–1593
- Grouiller F, Vercueil L, Krainik A, Segebarth C, Kahane P, David O (2007) A comparative study of different artefact removal algorithms for EEG signals acquired during functional MRI. *Neuroimage* 38:124–137
- Hu D, Yan L, Liu Y, Zhou Z, Friston KJ, Tan C, Wu D (2005) Unified SPM-ICA for fMRI analysis. *Neuroimage* 25:746–755
- Li Y-O, Adal T, Calhoun VD (2008) Estimating the number of independent components for functional magnetic resonance imaging data. *Hum Brain Mapp* 28:1251–1266
- Lutz A, Greischar LL, Rawlings NB, Ricard M, Davidson RJ (2004) Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. *Proc Natl Acad Sci USA* 101(46):16369–16373
- Makeig S, Debener S, Onton J, Delorme A (2004) Mining event-related brain dynamics. *Trends Cogn Sci* 8:204–210
- Mammone N, Morabito FC (2008) Enhanced automatic artifact detection based on independent component analysis and Renyi's entropy. *Neural Netw* 21:1029–1040
- McMenamin BW, Shackman AJ, Maxwell JS, Greischar LL, Davidson RJ. (in press). Validation of regression-based myogenic correction techniques for scalp and source-localized EEG. *Psychophysiology*
- Pizzagalli DA (2007) Electroencephalography and high-density electrophysiological source localization. In: Cacioppo JT, Tassinari LG, Berntson GG (eds) *Handbook of psychophysiology*. Cambridge University Press, NY, pp 56–84
- Seaman MA, Serlin RC (1998) Equivalence confidence intervals for two-group comparisons of means. *Psychol Methods* 3(4):403–411

- Tassinari LG, Cacioppo JT, Vanman EJ (2007) The skeletomotor system: surface electromyography. In: Cacioppo JT, Tassinari LG, Bertson GG (eds) *Handbook of psychophysiology*, 3rd edn. Cambridge University Press, NY, pp 267–302
- Whitham EM, Pope KJ, Fitzgibbon SP, Lewis T, Clark CR, Loveless S, Broberg M, Wallace A, DeLosAngeles D, Lillie P, Hardy A, Fronsco R, Pulbrook A, Willoughby JO (2007) Scalp electrical recording during paralysis: quantitative evidence that EEG frequencies above 20 Hz are contaminated by EMG. *Clin Neurophysiol* 118(8):1877–1888
- Whitham EM, Lewis T, Pope KJ, Fitzgibbon SP, Clark CR, Loveless S, DeLosAngeles D, Wallace AK, Broberg M, Willoughby JO (2008) Thinking activates EMG in scalp electrical recordings. *Clin Neurophysiol* 119(5):1166–1175

2 **Discussing Gamma**

3 **Christoph M. Michel · Micah M. Murray**

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6 This issue of Brain Topography contains a series of invited
7 short discussion articles on gamma-range high frequency
8 oscillations. A letter from Alexander J. Shackman, indi-
9 cating that one of a recently published article in Brain
10 Topography on frontal gamma oscillations did not consider
11 the possible contamination of facial EMG artifacts, initi-
12 ated this special topic. This was during the same time that
13 Shlomit Yuval-Greenberg et al. (2008) published a seminal
14 article demonstrating high correlation of miniature sac-
15 cades with transient induced gamma-band response
16 measured with the EEG. In this paper the authors also
17 demonstrated that proper inspection of the scalp topogra-
18 phy of the electric potential would most probably have
19 avoided the misinterpretation of previous data as occipital
20 gamma oscillations, since the potential map shows a very
21 strong gradient towards the nose reference. Since *Brain*
22 *Topography*, as the name indicates, is much concerned
23 with the proper analysis of the topography of the brain
24 electric/magnetic fields, we decided to ask some of the
25 most important groups in this ongoing discussion for a
26 short summary of their opinion. We are very happy that
27 they positively replied to this request and we are proud to
28 present their articles in this issue of *Brain Topography*.

29 The first article is written by Yuval-Greenberg and
30 Deouell (2009) from the Hebrew University of Jerusalem
31 in Israel. It is entitled “*The broadband transient induced*
32 *gamma-band response in EEG reflects an increase in the*
33 *rate of saccades*”. This article summarizes their findings on
34 the relation between saccades and induced Gamma activity
35 published in the paper mentioned above. It further stresses
36 the caution that has to be taken when interpreting induced

Gamma Band responses as neuronal synchronization. It 37
also points out that even source localization procedures 38
applied to EEG or MEG Gamma activity can be mislead- 39
ing, if the solution space of the head model is restricted to 40
the gray matter and excludes extra-cerebral sources. 41

Since these saccades are roughly time locked to the 42
stimulus and are appearing quasi after each stimulus, they 43
can neither be rejected nor averaged out. The authors thus 44
conclude that the only reasonable procedure to avoid 45
misinterpretation of such artifacts is to develop methods for 46
correction of the data. 47

Such correction algorithms are discussed in the second 48
article written by Shackman et al. (2009) from the Uni- 49
versity of Wisconsin in the USA. The article is entitled 50
“*Electromyogenic artifacts and electroencephalic infer-* 51
ences” and critically evaluates techniques designed to 52
separate neural signals from biological artifacts, in their 53
example electromyogenic activity. They particularly test 54
the performance of independent component analysis (ICA) 55
and convincingly demonstrate that ICA may not adequately 56
separate MEG from EEG signals in high-density 57
recordings. 58

The difficulty of properly removing myogenic artifacts 59
is particularly important for studies on EEG activity in the 60
Gamma range, given the fact that EMG activity has similar 61
spectral profiles. In addition, particularly facial EMG 62
activity can be sensitive to experimental manipulations in 63
the same way as neural Gamma activity. This has been 64
explicitly tested in a recent study on paralysed human 65
volunteers during the execution of different cognitive tasks 66
(Whitham et al. 2007). The results of these studies and new 67
data are reported in the article by Pope et al. (2009) from 68
the Flinders University in Adelaide, Australia. It is entitled: 69
“*Relation of gamma oscillations in scalp recordings to* 70
muscular activity.” While these studies clearly demonstrate 71

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72 a large contribution of muscle activity on the spectral
73 power and the coherence in unparalysed as compared to
74 paralysed subjects, it also shows that even in paralysed
75 subjects target stimuli still can induce enhanced EEG
76 activity in the gamma range, demonstrating that neuronal
77 gamma activity can still be measured with the scalp EEG.

78 While it is undisputable that scalp EEG and MEG is
79 largely contaminated by myogenic and ocular artifacts, it is
80 thought that this is much less the case for electrical activity
81 recorded intracranially. Such recordings can be performed
82 in patients undergoing invasive presurgical epilepsy eval-
83 uation. The group from the University Claude Bernard in
84 Lyon, France has repeatedly demonstrated intracranial
85 gamma-band oscillations related to different cognitive
86 tasks (e.g. Tallon-Baudry and Bertrand, 1999). In the
87 article by Jerbi et al. (2009) from Lyon and Paris, the group
88 critically evaluated the contribution of saccadic eye
89 movements to gamma-band power increase in intracranial
90 recordings. In this paper entitled “*saccade related gamma-*
91 *band responses in intracerebral EEG: dissociating neural*
92 *from ocular muscle activity*” the authors show that such
93 myogenic artifacts are picked up by intracranial electrodes
94 in the temporal pole because of its vicinity to extraocular
95 muscles. However, intracranial electrodes in other brain
96 structures are not influenced by activities of these muscles
97 and clearly show neuronal gamma activity related to cog-
98 nitive tasks.

99 Given the fact that neuronal Gamma activity indeed
100 exists (despite the difficulty to dissociate it from muscle
101 activity), the question remains on the functional signifi-
102 cance of these oscillations. Schroeder and Lakatos (2009)
103 from the Nathan S. Kline Institute for Psychiatry Research
104 in New York, USA, discuss this question in the article with
105 the provoking title “*The gamma oscillation: master or*
106 *slave*”. They point out the fact that gamma amplitude is
107 often coupled to the phase of lower frequency oscillations,
108 and that these lower frequency oscillations are often tuned
109 to the rhythm with which task-relevant stimuli are received
110 or sampled by motor behavior. The alignment of the brain
111 rhythm to the relevant stimuli permits optimal stimulus
112 discrimination during high excitability phases. It is during

these high excitability phases that gamma amplitude 113
increases. In consequence, gamma synchrony in theses 114
cases would only be secondary to the high excitability 115
phase that is determined by lower frequency rhythms, such 116
as alpha. In this sense, gamma might not necessary be the 117
rhythm that is critical for processing of task-relevant 118
stimuli. 119

All together, we think that these 5 special topic articles 120
on the generation and significance of gamma activity 121
highlight the major critical points of caution when 122
exploring high frequency oscillations with EEG and MEG 123
recordings. While there is no doubt that Gamma activity is 124
crucial for neuronal excitability, the exclusive role of this 125
rhythm for “binding” information in large-scale brain 126
networks may be doubtful given the obvious falsification of 127
some of the initial reports on induced gamma activity. 128
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References 130

- Jerbi K, Freyermuth S, Dalal S, Kahane P, Berthoz A, Bertrand O, Lachaux J-P (2009) Saccade related gamma-band responses in intracerebral EEG: dissociating neural from ocular muscle activity. *Brain Topogr* 131
132
133
134
Pope KJ, Fitzgibbon SP, Lewis TW, Whitham EM, Willoughby J (2009) Relation of gamma oscillations in scalp recordings to muscular activity. *Brain Topogr* 135
136
137
Schroeder C, Lakatos P (2009) The gamma oscillation: master or slave. *Brain Topogr* 138
139
Shackman AJ, McMenemy BW, Slagter HA, Maxwell JS, Greischar LL, Davidson RJ (2009) Electromyogenic artifacts and electroencephalic inferences. *Brain Topogr* 140
141
142
Whitham EM, Pope KJ, Fitzgibbon SP, Lewis T, Clark CR, Loveless S, Broberg M, Wallace A, De LosAngeles D, Lillie P, Hardy A, Fronsco R, Pulbrook A, Willoughby JO (2007) Scalp electrical recording during paralysis: quantitative evidence that EEG frequencies above 20 Hz are contaminated by EMG. *Clin Neurophysiol* 118:1877–1888 143
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Yuval-Greenberg S, Deouell L (2009) The broadband transient induced gamma-band response in EEG reflects an increase in the rate of saccades. *Brain Topogr* 149
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Yuval-Greenberg S, Tomer O, Keren AS, Nelken I, Deouell LY (2008) Transient induced gamma-band response in EEG as a manifestation of miniature saccades. *Neuron* 58:429–441 152
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